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ROTHWELL, FIGG, ERNST & MANBECK, P.C.
1425 K STREET, N.W.
SUITE 800
WASHINGTON, DC 20005

EXAMINER	
LU, FRANK WEI MIN	

ART UNIT	PAPER NUMBER
1634	

DATE MAILED: 02/14/2006

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/461,090	ULLRICH ET AL.	
	Examiner	Art Unit	
	Frank W Lu	1634	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *28 November 2005*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *39-48* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *39-48* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☒ All b)☐ Some * c)☐ None of:

1.☒ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

Response to Amendment

1. Applicant's response to the office action filed on November 28, 2005 has been entered. The claims pending in this application are claims 39-48. Rejection and/or objection not reiterated from the previous office action are hereby withdrawn in view of the response filed on November 28, 2005.

Claim Rejections - 35 USC § 112

2. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

3. Claims 39-43 and 45-48 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

Independent claims 39 and 45 have a limitation "G protein mediated extracellular signal transduction pathway". Although page 2, lines 5-22 of the specification describes that the activation of the growth-factor receptor is mediated by its extracellular domain and via an extracellular signal pathway, the specification fails to define or provide any disclosure to support such claim limitation. Furthermore, applicant does not indicate which part of the specification supports newly added claims 46 and 48.

MPEP 2163.06 notes "IF NEW MATTER IS ADDED TO THE CLAIMS, THE EXAMINER SHOULD REJECT THE CLAIMS UNDER 35 U.S.C. 112, FIRST PARAGRAPH - WRITTEN DESCRIPTION REQUIREMENT. *IN RE RASMUSSEN*, 650 F.2D 1212, 211 USPQ 323 (CCPA 1981)." MPEP 2163.02 teaches that "Whenever the issue arises, the fundamental factual inquiry is whether a claim defines an invention that is clearly conveyed to those skilled in the art at the time the application was filed...If a claim is amended to include subject matter, limitations, or terminology not present in the application as filed, involving a departure from, addition to, or deletion from the disclosure of the application as filed, the examiner should conclude that the claimed subject matter is not described in that application." MPEP 2163.06 further notes "WHEN AN AMENDMENT IS FILED IN REPLY TO AN OBJECTION OR REJECTION BASED ON 35 U.S.C. 112, FIRST PARAGRAPH, A STUDY OF THE ENTIRE APPLICATION IS OFTEN NECESSARY TO DETERMINE WHETHER OR NOT "NEW MATTER" IS INVOLVED. *APPLICANT SHOULD THEREFORE SPECIFICALLY POINT OUT THE SUPPORT FOR ANY AMENDMENTS MADE TO THE DISCLOSURE*" (emphasis added).

Response to Arguments

In page 6, second paragraph of applicant's remarks, applicant argues that "[C]laims 39-43 and 45 were rejected under 35 USC § 112, first paragraph, as lacking an adequate written description for the limitation 'G protein mediated extracellular signal transduction pathway'. This limitation is supported by the disclosure on page 2, lines 5-22 of the present application".

This argument has been fully considered but it is not persuasive toward the withdrawal of the rejection. Although page 2, lines 5-22 of the specification describes that the activation of the growth-factor receptor is mediated by its extracellular domain and via an extracellular signal pathway, the specification does not describe the phrase "G protein mediated extracellular signal transduction pathway".

Claim Rejections - 35 USC § 102

4. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless –

(a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.

5. Claims 39, 40, 42-45, and 47 are rejected under 35 U.S.C. 102(a) as being anticipated by Dong *et al.*, (Proc. Natl. Acad. Sci. USA, 96, 6235-6240, May 1999).

Dong *et al.*, teach metalloprotease-mediated ligand release regulates autocrine signaling through the epidermal growth factor receptor.

Regarding claims 39, 40, 42, 43, and 47, since Dong *et al.*, teach to incubate HMEC cells with batimastat or antagonist mAb225 for 24 hr and then treat the HMEC cells with EGF for 20 min (see page 6238, right column and Figure 4) and teach that ligands such as EGF that activate the epidermal growth factor receptor (EGFR) are synthesized as membrane-anchored precursors that are proteolytically released by members of the ADAM family of metalloproteases and batimastat is a metalloproteinase inhibitor that prevents EGFR ligand such as EGF release by abolish biological activity of the metalloproteinases (see page 6235, abstract and right column, and page 6239, right column, last paragraph), and there is no evidence to show that batimastat can not affect a G protein or G protein coupled receptor initiated extracellular signal pathway wherein said G protein mediated extracellular signal transduction pathway includes cleavage of a growth factor receptor and claims 39 and 47 do not require that stimulating step must be performed before contacting step and the phrase "wherein said G protein mediated extracellular signal transduction pathway includes cleavage of a growth factor receptor" is not a method step of claims 39 and 47, Dong *et al.*, disclose contacting a cell with a compound (ie., batimastat) which indirectly acts on a growth factor precursor (by preventing EGFR ligand such as EGF release) in a G protein mediated extracellular signal pathway as recited in claims 39 and 47. Since Dong *et al.*, teach that the inhibitory effect of batimastat on EGFR tyrosine

phosphorylation of the HMEC cells is totally reversed by EGF (see Figure 4, column 5 in the presence of batimastat +EGF), batimastat has no effect on EGFR tyrosine phosphorylation of HMEC cells in the presence of EGF, comparing with batimastat treated HMEC cells, the HMEC cells treated with batimastat +EGF has an increased level of EGFR tyrosine phosphorylation (see page 6238, right column and Figure 4). Since it is known that increased level of EGFR tyrosine phosphorylation in a cell indicates that a G protein or G protein coupled receptor initiated extracellular signal pathway of the cell has been activated (for evidence, see canceled claim 36 of this instant application), Dong *et al.,* disclose stimulating G protein mediated signal transduction in a cell (ie., treating the HMEC cells with batimastat+EGF) having a receptor tyrosine kinase (ie., EGFR) wherein the receptor tyrosine kinase is activated and thereby modulating the receptor tyrosine kinase activation by G-protein-mediated signal transduction (ie., increasing the level of EGFR tyrosine phosphorylation) as recited in claims 39 and 47 wherein said tyrosine kinase is EGFR as recited in claims 40, 42, and 43.

Regarding claim 44, Dong *et al.,* teach to incubate HMEC cells with batimastat or antagonist mAb225 for 24 hr and then treat the HMEC cells with EGF for 20 min (see page 6238, right column and Figure 4). Since it is known that increased level of EGFR tyrosine phosphorylation in a cell indicates that a G protein or G protein coupled receptor initiated extracellular signal pathway of the cell has been activated (for evidence, see canceled claim 36 of this instant application) and Dong *et al.,* teach that batimastat decreases level of EGFR tyrosine phosphorylation in the HMEC cells (see page 6238, right column and Figure 4), Dong *et al.,* disclose contacting a cell containing a receptor tyrosine kinase (ie., a HMEC cell) capable of activation by G-protein mediated signal transduction with a test compound (ie., batimastat) as

recited in the claim. Since Dong *et al.,* teach that batimastat is a selective metalloprotease inhibitor that prevents EGFR ligand release (see page 6235, abstract and right column, and page 6239, right column, last paragraph) and there is no evidence to show that batimastat can not affect a G protein or G protein coupled receptor initiated extracellular signal pathway, Dong *et al.,* disclose a test compound (ie., batimastat) suspected to indirectly act on a ligand precursor of the receptor tyrosine kinase (ie., EGFR, by preventing EGFR ligand release) as recited in the claim. Since Dong *et al.,* teach to compare the level of EGFR tyrosine phosphorylation of the HMEC in the presence of batimastat, antagonist mAb225 or EGF (see Figure 4), Dong *et al.,* disclose evaluating G-protein mediated receptor tyrosine kinase (ie., EGFR) activation upon exposure of the cell (ie., the HMEC cells) to said test compound (ie., batimastat) as an indication of said test compound's ability (ie., with or without ability) to modulate G-protein mediated signal transduction thereby identifying a test compound for modulating G-protein mediated signal transduction as recited in the claim.

Regarding claim 45, since Dong *et al.,* teach to incubate HMEC cells with batimastat or antagonist mAb225 for 24 hr and then treat the HMEC cells with EGF for 20 min (see page 6238, right column and Figure 4) and teach that ligands such as EGF that activate the epidermal growth factor receptor (EGFR) are synthesized as membrane-anchored precursors that are proteolytically released by members of the ADAM family of metalloproteases and batimastat is a metalloproteinase inhibitor that prevents EGFR ligand such as EGF release by abolish biological activity of the metalloproteinases (see page 6235, abstract and right column, and page 6239, right column, last paragraph), and there is no evidence to show that batimastat can not affect a G protein or G protein coupled receptor initiated extracellular signal pathway and claim 45 does not

require that stimulating step must be performed before contacting step, Dong *et al.,* disclose contacting a cell with a compound (ie., batimastat) which indirectly acts on a growth factor precursor (by preventing EGFR ligand such as EGF release) in a G protein mediated extracellular signal pathway as recited in claim 45. Since Dong *et al.,* teach that the inhibitory effect of batimastat on EGFR tyrosine phosphorylation of the HMEC cells is totally reversed by EGF (see Figure 4, column 5 in the presence of batimastat +EGF), batimastat has no effect on EGFR tyrosine phosphorylation of HMEC cells in the presence of EGF, comparing with batimastat treated HMEC cells, the HMEC cells treated with batimastat +EGF has an increased level of EGFR tyrosine phosphorylation (see page 6238, right column and Figure 4). Since it is known that increased level of EGFR tyrosine phosphorylation in a cell indicates that a G protein or G protein coupled receptor initiated extracellular signal pathway of the cell has been activated (for evidence, see canceled claim 36 of this instant application), Dong *et al.,* disclose stimulating G protein mediated signal transduction in a cell (ie., treating the HMEC cells with batimastat+EGF) having a receptor tyrosine kinase (ie., EGFR) wherein the receptor tyrosine kinase is activated and thereby modulating the receptor tyrosine kinase activation by G-protein-mediated signal transduction (ie., increasing the level of EGFR tyrosine phosphorylation) wherein said tyrosine kinase is EGFR as recited in claim 45. Since it is known that EGFR has an extracellular domain and a cell comprising EGFR has a G-protein mediated signal transduction pathway wherein EGFR activation occurs by tyrosine phosphorylation of EGFR (see the specification, page 1, last paragraph, and page 2, second paragraph), Dong *et al.,* disclose that said receptor tyrosine kinase is EGFR and said cell (ie., the HMEC cell) comprising the extracellular domain of EGFR and having a G-protein mediated signal transduction pathway

wherein one or more tyrosine residues are phosphorylated based on the activation of said G-protein mediated signal transduction pathway as recited in claim 45. Since Dong *et al.,* teach that EGF is generated from its membrane-anchored precursor by one of the ADAM family of metalloproteases (see page 6235, abstract) and it is know that EGF binds to the extracellular domain of EGFR, Dong *et al.,* disclose that the extracellular domain of said receptor (ie., EGFR) is capable of binding to its receptor ligand (ie., EGF) and said ligand is generated from a precursor of said ligand (ie., the precursor of EGF) by a proteinase-dependent cleavage (ie., one of the ADAM family of metalloproteases) thereby modulating the receptor tyrosine kinase activation by G-protein mediated signal transduction as recited in claim 45.

Therefore, Dong *et al.,* teach all limitations recited in claims 39, 40, 42-45 and 47.

Response to Arguments

In page 6, fourth paragraph bridging to page 7, first paragraph of applicant's remarks, applicant argues that "[I]n the presently claimed method, the modulator binds directly to the growth factor receptor. In contrast to the present invention, Dong uses batimastat which inhibits the metallo-proteinase. Thus, the present invention inhibits receptor tyrosine kinase transactivation by a different mechanism. For example, the recognition sequence for a metalloproteinase may be masked by binding of the modulator in the present method or the binding of the modulator may inhibit the binding of the growth factor to the receptor. In Dong, the enzymatic activity of the metalloproteinase is inhibited using batimastat. Though Dong's method can interrupt the whole signal cascade, the present invention interrupts the signal cascade in a different way. In the present invention a compound binds to the growth factor

precursor inhibiting processing of the precursor and interrupting the signal cascade. Thus, the present inventors have shown for the first time that modulators which act on a growth factor precursor to inhibit the activation of the extracellular domain of a growth factor receptor are suitable for the treatment of disorders, in particular of cancers, which are induced by G- protein mediated signal transduction. In contrast to the present invention, Dong discloses only that the inhibitory effect of batimastat on metastasis is due to interference with autocrine EGFR signaling. Thus, Dong does not suggest or disclose a method for identifying and providing modulators according to the present invention

These arguments have been fully considered but they are not persuasive toward the withdrawal of the rejection. First, the claims do not require that the modulator binds directly to the growth factor receptor as suggested by applicant. Second, the claims do not require that a compound directly binds to the growth factor precursor inhibiting processing of the precursor and interrupting the signal cascade as suggested by applicant. Third, Dong *et al.*, teach to indirectly act on a growth factor precursor by preventing EGFR ligand such as EGF release using batimastat.

Claim Rejections - 35 USC § 103

6. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

7. Claim 41 is rejected under 35 U.S.C. 103(a) as being unpatentable over Dong *et al.,* (May, 1999) as applied to claims 39, 40, 42-45, and 47 above, and further in view of Miyoshi *et al.,* (J. Biol. Chem., 272, 14349-14355, 1997).

The teachings of Dong *et al.,* have been summarized previously, *supra.*

Dong *et al.,* do not disclose that said precursor of the ligand for the receptor tyrosine kinase is proHB-EGF as recited in claim 41.

Miyoshi *et al.,* do teach a cell line, AH66tc, that can produce proHB-EGF and contains EGFR (see abstract in page 14349, right column in page 14351, and Figure 4 in page 14352).

Therefore, it would have been *prima facie* obvious to one having ordinary skill in the art at the time the invention was made to have used AH66tc to perform the method recited in claim 39 in view of the references of Dong *et al.,* and Miyoshi *et al.,* so that HB-EGF released from pro-HB-EGF can activate EGFR by binding to its extracellular domain. One having ordinary skill in the art would have been motivated to do so because the simple replacement of one kind of cell line that is capable to produce a ligand of EGFR (ie., a human mammary epithelial cell line that can produce EGF taught by Dong *et al.,*) from another kind of cell line that is capable to

produce a ligand of EGFR (ie., AH66tc that can produce HB-EGF taught by Miyoshi *et al.*,) during the process of performing the method recited in claim 41 would have been, in the absence of convincing evidence to the contrary, *prima facie* obvious to one having ordinary skill in the art at the time the invention was made because the replacement would not change the method steps of claim 41 since it is known that a variety of ligands such as HB-EGF in addition to EGF have been shown to stimulate EGFR and is released from their membrane-anchored precursors (see Dong *et al.*, page 6235, left column).

Furthermore, the motivation to make the substitution cited above arises from the expectation that the prior art elements will perform their expected functions to achieve their expected results when combined for their common known purpose. Support for making the obviousness rejection comes from the M.P.E.P. at 2144.06, 2144.07 and 2144.09.

Also note that there is no invention involved in combining old elements is such a manner that these elements perform in combination the same function as set forth in the prior art without giving unobvious or unexpected results. *In re Rose* 220 F.2d. 459, 105 USPQ 237 (CCPA 1955).

Response to Arguments

In page 7, last paragraph bridging to page 8, first paragraph of applicant's remarks, applicant argues that "[M]iyoshi was cited for the disclosure of a cell line which produces ProHB-EGF and contains EGFR. Miyoshi does not suggest or disclose contacting the cell with a compound which binds to a growth factor precursor in a G protein mediated extracellular signal transduction pathway wherein said G protein mediated extracellular signal transduction pathway includes cleavage of a growth factor receptor, or a step of stimulating the G protein/GPCR initiated signal transduction pathway as required in the first step of the present claims followed

by a second step of contacting a cell with a compound affecting a G protein mediated extracellular signal transduction pathway and thus does not cure the above discussed deficiencies in Dong".

These arguments have been fully considered but they are not persuasive toward the withdrawal of the rejection. First, the claims do not require contacting the cell with a compound which binds to a growth factor precursor as suggested by applicant. Second, Dong *et al.,* do teach a step of stimulating the G protein/GPCR initiated signal transduction pathway as required in the first step of the present claims followed by a second step of contacting a cell with a compound affecting a G protein mediated extracellular signal transduction pathway (see above rejection under 35 U.S,C 102).

8. Claims 46 and 48 are rejected under 35 U.S.C. 103(a) as being unpatentable over Dong *et al.,* (May, 1999) as applied to claims 39, 40, 42-45, and 47 above, and further in view of Sherwood *et al.,* (British Journal of Cancer, 77, 855-861, March 1998).

The teachings of Dong *et al.,* have been summarized previously, *supra.*

Dong *et al.,* do not disclose that said cell is an ovarian cancer cells or a prostate cancer cell as recited in claims 46 and 48.

Sherwood *et al.,* teach several prostate cancer cells having a high expression of EGF receptor such as PC3 and DU145 (see abstract in page 855).

Therefore, it would have been *prima facie* obvious to one having ordinary skill in the art at the time the invention was made to have performed the method recited in claim 46 or 48 wherein said cell is a prostate cancer cell (ie., PC3 cell line) in view of the references of Dong *et*

al., and Sherwood *et al.*. One having ordinary skill in the art would have been motivated to do so because the simple replacement of one kind of cell line having a receptor tyrosine kinase (ie., the cell line having EGF receptor taught by Dong *et al.*,) from another kind of cell line having a receptor tyrosine kinase (ie., the cell line having EGF receptor such as PC3 taught by Sherwood *et al.*,) during the process of performing the method recited in claim 45 or 48 would have been, in the absence of convincing evidence to the contrary, *prima facie* obvious to one having ordinary skill in the art at the time the invention was made because the cell line having EGF receptor taught by Dong *et al.*, and the cell line having EGF receptor such as PC3 taught by Sherwood *et al.*, are used for the same purpose (ie., measuring activation of EGF receptor).

Furthermore, the motivation to make the substitution cited above arises from the expectation that the prior art elements will perform their expected functions to achieve their expected results when combined for their common known purpose. Support for making the obviousness rejection comes from the M.P.E.P. at 2144.06, 2144.07 and 2144.09.

Also note that there is no invention involved in combining old elements is such a manner that these elements perform in combination the same function as set forth in the prior art without giving unobvious or unexpected results. *In re Rose* 220 F.2d. 459, 105 USPQ 237 (CCPA 1955).

Conclusion

9. Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

10. No claim is allowed.

11. Papers related to this application may be submitted to Group 1600 by facsimile transmission. Papers should be faxed to Group 1600 via the PTO Fax Center. The faxing of such papers must conform with the notices published in the Official Gazette, 1096 OG 30 (November 15, 1988), 1156 OG 61 (November 16, 1993), and 1157 OG 94 (December 28, 1993)(See 37 CAR § 1.6(d)). The CM Fax Center number is (571)273-8300.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Frank Lu, Ph.D., whose telephone number is (571)272-0746. The examiner can normally be reached on Monday-Friday from 9 A.M. to 5 P.M.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, W. Gary Jones, can be reached on (571)272-0745.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.

Frank Lu
Primary Examiner
February 7, 2006

FRANK LU
Primary PATENT EXAMINER